Postconcussional Disorder and Loss of Consciousness

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Postconcussional disorder (PCD) has been described in the psychiatric, neurological, neuropsychological, and rehabilitation medicine literature for many years. PCD has recently been introduced into DSM-IV, appearing in an appendix that contains a number of proposals for new categories and axes that were suggested for possible inclusion in DSM-IV. There are some major difficulties with the proposed criteria for PCD. This article explores some of these difficulties, particularly focusing on the criteria of loss of consciousness (LOC). A review of the literature demonstrates that LOC is not necessary for PCD to occur. The major difficulty with the DSM-IV criteria is the definition of concussion. The article suggests that, instead, the criteria for mild traumatic brain injury, as defined by the American Congress of Rehabilitation Medicine, may be more appropriate.

Postconcussional disorder (PCD) has been described in the medical literature for over a century. The term Post-concussion syndrome was coined by Strauss and Savitsky in 1934.[1] PCD is the most prevalent and yet controversial neuropsychiatric diagnosis following brain injury. PCD is linked most commonly to minor brain injury, because the symptoms are unobscured by the myriad of findings that accompany a more severe brain injury. The constellation of symptoms includes physical symptoms, cognitive deficits, and emotional sequelae. PCD is described in the neurological, neuropsychological, psychiatric, and rehabilitation medicine literature. Common physical symptoms include headache pain, nausea, dizziness or vertigo, unsteadiness or poor coordination, tinnitus, hearing loss, blurred vision, diplopia, convergence insufficiency, light and noise sensitivity, and altered sense of taste and smell. The cognitive deficits include memory difficulties, decreased attention and concentration, decreased speed of information processing, communication difficulties, difficulties with executive functioning (including initiation and planning, judgment and perception), and an increased sensitivity to lack of sleep, fatigue, stress, drugs, and alcohol. Emotional symptoms include emotional lability, irritability and aggression, a change in personality, fatigue and decreased energy, anxiety, depression, apathy, disordered sleep, loss of libido, and poor appetite.[2]

Although the underlying pathology of PCD is uncertain, a generally accepted theory is that it is caused by rotational

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sheer strains and corresponding diffuse axonal injury throughout the brain. Studies of primates have confirmed that acceleration of the head without impact can cause severe diffuse destruction of brain substance. Gennarelli *et al.*[3] produced traumatic coma in 45 monkeys by accelerating their heads without impact. At autopsy, the principal abnormality seen was best appreciated microscopically and consisted of diffuse axonal injury (DAI), which manifest as axonal retraction balls or abnormalities of axonal morphology in the white matter of the brain. Axonal damage was not confined to focal areas, but rather was scattered widely throughout the white matter of the cerebral hemispheres. Oppenheimer[4] found damaged axons with neuropathological changes similar to more severe injury in five patients with mild traumatic brain injury who had died from other injuries. One of the patients had been knocked down by a motor scooter, had no loss of consciousness, and was described only as "stunned" following his accident.

In addition to DAI, focal injuries may occur following head injury. For example, contusions may appear on the undersurface of the temporal and frontal lobes and the anterior pole of the temporal lobes due to contact with rough bony surfaces. The orbital frontal cortex is particularly sensitive to damage during acceleration/deceleration injuries because of its proximity to the bony structures of the skull. This area is sometimes irreverently referred to as the "dashboard" of the brain.[5] Many patients even with severe focal contusions never lose consciousness. In the 1984 *Neurology and Neurosurgery Update Series*, Alexander[6] included a computed tomography (CT) scan of a 59-year-old man who never lost consciousness, but developed amnesia and a personality change. His CT scan clearly demonstrated hemorrhagic lesions in anterior and inferior frontal regions.

Patients suffering from PCD usually have a reduction in the overall speed, efficiency, execution, and integration of mental processes. This has been described as "reduced information processing capacity" by Gronwall.[7] Following mild brain injury, patients have difficulties in areas that require them to analyze complex information, and they therefore present as slower, more distractible, and forgetful. When patients are concentrating on point A, they are unable to also process point B simultaneously, and they therefore present as inattentive because they are unable to process a normal stream of information.

Often there are few if any findings on physical examination, and the microscopic brain damage is usually not detected with conventional imaging techniques such as CT or magnetic resonance imaging (MRI) scanning. Neuropsychological assessment is often undertaken, but tends to err in the direction of underestimating disorders. Many valid problems are not registered on neuropsychological testing. Orbital-frontal deficits are difficult to detect with standard neuropsychological testing. Subtle changes in attention and concentration, new learning ability, word retrieval, and judgment often do not register in the testing. Traditional IQ tests are often insensitive. Although no single test is diagnostic of

frontal lobe functioning, the tinker toy test has been shown to have some predictive value with regard to future employment.[8]

Most available tests of memory assess verbal memory and learning and do not involve high level processing of complex information. Patients, however, often complain of difficulties with episodic memory (for day to day activities) or with procedural memory (the learning and recall process). It is important, therefore, to carefully interview patients and obtain collateral information from family, friends, and employers. Psychiatric and neuropsychological assessments are performed in quiet, controlled environments that do not include the distractions and frustrations of everyday living, and therefore difficulties may be underestimated.

Testing of a patient within a few months of the injury with repeated testing a year or more later may provide more useful information then an examination given at only one point in time. Testing that reveals improvement over time helps confirm that the disorder began at the time of the brain injury.[9]

Fortunately, the majority of studies suggest that although PCD is seen in the majority of patients within the first month following mild traumatic brain injury, the incidence of PCD is reduced significantly by three to six months following the injury.[10] However, at one year after injury, approximately 15 percent of patients still have disabling symptoms.[11, 12] Patients at high risk of having persistent PCD symptoms include those with a history of head injury and older patients (probably above 40 years of age).[13] Although the cognitive deficits may be temporary, this does not mean that brain damage is reversible. Brain tissue does not regenerate. The cumulative effect of subsequent head injury, causing ongoing cognitive deficits, is an example of the ongoing residual effects of brain injury. As discussed by Gronwell,[7] there may be persistent "cognitive fragility" to central nervous system (CNS) stressors. Ewing *et al.*[14] compared performance under conditions of mild hypoxia in a group of university students who had made a "full recovery" from mild head injury between one and three years before the study was done with a matched group of control students who had never had a head injury. The mild head injury group performed, when mildly hypoxic, at a significantly lower level than control subjects on a memory and vigilance task. Although the students who had suffered previously from mild head injury had returned to their previous level of functioning and had engaged in full-time university work, the mild head injury may have left a residual effect that impaired their ability to withstand another CNS stressor.

Psychological factors may also influence late symptoms. This often occurs when primary deficits are undiagnosed, resulting in a dysfunctional cycle. Patients are frequently bewildered and overwhelmed by their symptoms. Despite having relatively mild injuries, they may continue to be plagued by problems such as headaches, lack of energy, dizziness, and an inability to concentrate on or cope with life's stressors. It is not surprising, therefore, that patients become frustrated, angry, and depressed. Kay[15] described how a person's sense of predictability and

Table 1
DSM IV Research Criteria for PCD[a]

A. A history of head trauma that has caused significant cerebral concussion.

B. Evidence from neuropsychological testings or quantified cognitive assessment of difficulty in attention or memory.

C. Three or more of the following occur shortly after the trauma and last at least three months:
   1. becoming fatigued easily;
   2. disordered sleep;
   3. headache;
   4. vertigo or dizziness;
   5. irritability or aggression on little or no provocation;
   6. anxiety, depression, or affective lability;
   7. changes in personality;
   8. apathy or lack of spontaneity.

D. The symptoms in criteria B and C have their onset following head trauma or else represent a substantial worsening of preexisting symptoms.

E. The disturbance causes significant impairment in social or occupational functioning and represents a significant decline from a previous level of functioning.

F. The symptoms do not meet the criteria for dementia due to head trauma and are not better accounted for by another mental disorder.

[a]See Reference 17.

stability may be disrupted, especially in the absence of external validation of symptoms; a cycle of "failure, fear, avoidance, anxiety, depression, loss of self-esteem, isolation, and alienation" may result. Personality changes and psychiatric symptoms such as irritability may occur as a direct result of mild traumatic brain injury, but depressive symptoms tend not to occur until at least six months after the injury.[16]

DSM IV Criteria for PCD

The research criteria for PCD, as described in DSM IV, is given in Table 1. PCD is listed in Appendix B of DSM-IV, which provides criteria sets and axes for further study.[17] In the text of DSM-IV, it is noted that there is insufficient evidence to establish a definite threshold for the severity of closed head injury, but specific criteria are suggested as including two of the following: (1) a period of unconsciousness lasting more than five minutes; (2) a period of posttraumatic amnesia that lasts more than 12 hours after closed head injury; or (3) a new onset of seizures (or marked worsening of a preexisting seizure disorder) that occurs within the first six months after the closed head injury.

There are several difficulties with the definition of PCD provided in DSM IV. The bulk of this article will focus on the criteria of loss of consciousness (LOC). Other apparent difficulties include the DSM-IV description of how PCD occurs as a consequence of closed head injury. However, "head injury" does not need to occur in order for brain damage to occur. Head injury is a poorly defined term that refers to an injury to the head, face, and

Table 2
Definition of Mild Traumatic Brain Injury[a]

A patient with MTBI is a person who has had traumatically induced physiological disruption of brain function, as manifested by at least one of the following:

1. any period of loss consciousness;
2. any loss of memory for events immediately before or after the accident;
3. any alteration in mental state at the time of the accident (e.g., feeling dazed, disoriented, or confused); and
4. focal neurological deficit(s) that may or may not be transient; but where the severity of the injury does not exceed the following:
    1. loss of consciousness of approximately 30 minutes or less;
    2. after 30 minutes, an initial Glasgow Coma Scale (GCS) of 13–15; and
    3. posttraumatic amnesia (PTA) not greater than 24 hours.

[a] From the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine.[35]

neck area, which does not necessarily cause injury to the brain. Instead, a more appropriate term would be "brain injury." The definition of mild traumatic brain injury (MTBI) is fully defined later in this article (see Table 2).

Because seizures rarely occur following MTBI, the proposed DSM-IV threshold criteria for concussion would essentially require both a period of LOC longer than five minutes as well as posttraumatic amnesia (PTA) lasting longer than 12 hours. One difficulty is that PTA is not adequately defined. A generally accepted definition of PTA is the interval between the injury and the time when the patient begins to lay down continuous memory of ongoing events. PTA includes the time during which the patient was awake, but confused. Patients need to be carefully questioned, because approximately one-third of patients who are questioned following MTBI give a history of an island of recall well before memories become continuous, resulting in a potential error of underestimating the total duration of PTA.[18] Patients with MTBI are often discharged from the hospital while still suffering PTA. When patients are interviewed retrospectively, it cannot be assumed that their memory is reliable, especially if they have had repeated briefings by family members and others. Prospective assessment that commences prior to the resolution of PTA overcomes the possible errors inherent in assessing PTA retrospectively by clinical interview, but this is not always possible.[19]

The duration of PTA has been used as an indicator of the severity of injury. In 1932, Russell proposed mild head injury as causing PTA lasting up to 1 hour, moderate head injury as causing PTA lasting longer than 1 hour but less than 24 hours, and severe head injury as causing PTA lasting longer than 24 hours.[20] Using a duration of 12 hours of PTA as a cut-off for PCD would therefore rule out a number of patients who are suffering from moderate head injury, who have PTA less than 12 hours, and would by definition exclude all patients with mild head injury.

Another difficulty with the DSM-IV criteria is the stipulation that symptoms need to be present for at least three months following the trauma. Evidence in the literature has suggested that for most patients PCD symptoms are reduced significantly by three months posttrauma.

One study conducted at three different locations showed that at one month after the injury most patients demonstrated attentional deficits and reduced visual motor speed. These problems, and associated complaints of headache, fatigue, and dizziness, diminished significantly during the next two months. However, at three months almost all of the patients still complained of headaches and many of them complained of fatigue and dizziness.[21] Some patients may only have marked impairment in their ability to function at work for a few weeks following mild traumatic brain injury, and it is unclear why the DSM IV criteria requires a duration of symptoms for at least three months.

Concerning the symptoms themselves, DSM-IV includes a number of physical, cognitive, and emotional symptoms. Physical symptoms such as difficulties with headaches, dizziness and vertigo, hearing loss, visual problems, and diminished taste and smell are discussed. However, it is unclear why other physical symptoms such as nausea, tinnitus, impaired coordination and balance, and light and noise sensitivity are not included. These physical symptoms commonly occur following MTBI, and some of them may cause a marked impairment in the ability to return to work. It is also unclear why the DSM-IV criteria only include the cognitive deficits of memory and attention and concentration. Most patients also have impairment in their speed of information processing as represented with testing such as the paced auditory serial addition task (PASAT).[22] Patients also have difficulties with executive functioning, which includes setting goals, assessing strengths and weaknesses, planning and directing activity, initiating and inhibiting behavior, monitoring activity, and evaluating results. There may also be difficulties with communication, including inefficient word retrieval, tangentiality of thought and speech, overtalkativeness, use of peculiar words and phrases, and uninhibited choice of words. There may also be disorders of judgment and perception, including misinterpretation of actions or intentions of others, a tendency to be socially inappropriate in verbal communications, and unrealistic appraisal of onesself and one's strengths and weaknesses. Finally, there may be increased sensitivity to lack of sleep, fatigue, stress, drugs, and alcohol.[2] It is unclear why the DSM-IV criteria include only memory, attention, and concentration difficulties and do not include these other cognitive deficits that commonly occur following MTBI.

According to the DSM-IV criteria, if a patient has difficulties with memory functioning and executive functioning following mild traumatic brain injury, he or she should be given a diagnosis of dementia, not a diagnosis of PCD.[23] In many ways, a diagnosis of dementia is easier to make than a diagnosis of PCD, which is counterintuitive to what one would expect. Following MTBI, a patient need only demonstrate problems with executive functioning and memory to be given a diagnosis of dementia. There is no need for the patient to experience a period of LOC or PTA to be given a diagnosis of dementia.

One of the major difficulties with the

DSM-IV criteria is not only that LOC of longer than five minutes is suggested, but also that the term "loss of consciousness" (or LOC) has not been adequately defined. Consciousness is not an "all or nothing" phenomenon. Instead, there are levels of consciousness. For example, if an accident victim is able to respond to painful stimuli but not verbal stimuli, are they then labeled as being unconscious or conscious? The answer is unclear. Definitions of consciousness include vigilance, the ability to react to the environment, and awareness of facts and the content of mental phenomenon.[24] Certain terms have been used to describe degrees of consciousness including "alert wakefulness, lethargy, obtundation, stupor, and coma." However, these terms have not been adequately defined, and it is unclear from the DSM IV criteria whether LOC refers, for example, to "obtundation" or to "stupor," to both or to neither. There are difficulties, therefore, in defining consciousness or loss of consciousness. It is assumed that others use the same definition of LOC, but this may not be the case.

The Glasgow Coma Scale (GCS), often used by paramedics as well as physicians, may provide a more accurate, objective tool for measuring levels of consciousness. It should be noted, however, that the GCS was designed to classify severe brain injuries and may be of little relevance for patients suffering from mild traumatic brain injury. Brian Jennett,[25] the originator of the GCS, described how the GCS was not intended to be a means of distinguishing among different types of milder injuries. Jennett wrote that "many of these patients are oriented by the time they are first assessed and therefore score at the top of the Glasgow Scale. Yet some of these patients have had a period of altered consciousness, either witnessed or evidenced by their being amnesic for events immediately following injury. The impairment of consciousness is indicative of diffuse brain damage, but there can also be marked local damage without either alteration in consciousness or amnesia."[25]

Research on LOC and PCD

Despite the difficulties in defining LOC, following are summaries of a few studies that explore the issue of LOC and PCD.

Yarnell and Rossie[26] investigated 27 patients who suffered apparent whiplash injuries in motor vehicle accidents. The patients were assessed at least one year following their injuries. None of the patients were more than initially dazed from their accidents, and periods of PTA were brief. All of the patients had been employed prior to their accidents. The patients suffered from the classical symptoms of PCD. Neurological examination and neurodiagnostic testing (MRI, electroencephalogram, and brain stem auditory response testing) results were essentially normal. However, neuropsychological evaluation showed impairments on tests of vigilance, selective attention, memory, mental stamina, and cognitive flexibility. At 18 months postinjury, none of the patients tested had returned to their previous level of occupational functioning. Fifty percent of them were unemployed and the other 50

percent were working at a reduced capacity in terms of hours or income level. Active involvement in a lawsuit did not correlate with return to work.

In a commonly quoted study, Lenniger *et al.*[27] examined 53 patients who had experienced PCD symptoms for at least one month following motor vehicle accidents. Thirty-one patients had sustained a brief LOC (concussion group). Twenty-two were only dazed, with no LOC (mild concussion group). Eight neuropsychological tests were selected for their ability to measure brain dysfunction. The results demonstrated that mild head injury patients compared with control subjects experienced deficits on test of reasoning (category test), information processing (PASAT-revised), verbal learning (auditory verbal learning test), and attention and organization (complex figure-copy). There was no evidence that injuries associated with LOC were more debilitating than injuries that resulted in "dazing" but no LOC. In other words, as far as minor head injuries are concerned, the study showed that the occurrence of LOC did not distinguish people as being at greater risk for neuropsychological consequences. In addition, the authors found no evidence of differences between the litigating and the nonlitigating patients in their study.

One difficulty with such studies is that neuropsychological testing is not completely objective. Any examination that allows a patient to give at least two possible answers is not completely objective. However, in recent years there have been objective studies of brain functioning using imaging techniques such as positron emission tomography (PET). PET scanning is a computerized technique that produces a picture showing the distribution of radioactivity in the brain, after the injection of a radioactive isotope. Whereas a CT or MRI scan provides a static picture of brain structure, the PET scan reflects brain function by showing blood flow and metabolic activity in different areas of the brain. It provides an illustration of disruptions in brain metabolism by monitoring the amount of glucose that brain cells consume. Humayun *et al.*[28] studied regional glucose utilization with PET in three patients with mild head injury and persisting cognitive deficits. All three patients had normal MRI and CT scans. Compared with three control subjects, the patients had abnormal glucose utilization in several frontal and temporal regions and the left caudate nucleus.

In a recent study by Ruff *et al.*,[29] nine MTBI cases were examined. Four of the nine patients reported no LOC. A control group of 24 right-handed volunteers was used. All nine patients had negative CT or MRI findings, but had positive neuropsychological results. Patients had impairment on tests of sensory-motor functioning, attention, memory and learning, language, spacial abilities, as well as problem-solving. All subjects received the continuous performance test (CPT), for which they were instructed during uptake. The PET findings validated the neurobehavioral sequelae on an objective basis not only for those with, but also for those without, LOC. The patients were examined at an average of 18 months postaccident. Neither the neuropsychological findings nor PET findings were

substantially different between mildly injured patients with or without LOC. The PET procedures documented neuropathology, which was frequently pronounced in the frontal and anteriotemporal regions.

Although further study is needed, it is clear that both PET and neuropsychological test results provide converging evidence that even mild traumatic brain injury without LOC may lead to neuropathology in the absence of evidence from MRI or CT scanning. Functional imaging techniques such as functional MRI, PET, and single photon emission computerized tomography (SPECT) are, however, mainly research tools at the present time and further studies are needed to determine their validity.

General Literature on LOC and PCD

In addition to clinical research, it has generally been accepted in the scientific literature that LOC is not necessary for postconcussional disorder to occur. Textbooks and review articles in the neurological, neuropsychological, psychiatric, and rehabilitation medicine literature have generally adopted the opinion that MTBI or PCD may occur without LOC. For example, in *Prognosis of Neurological Disorders*, Evans[30] wrote that LOC does not have to occur for the postconcussion syndrome to develop. In *Current Therapy in Neurological Disease*, Gordon[31] described how no LOC or temporary lapse of cerebral function was necessary. Gordon wrote that "Any sufficient blow, fall, or acceleration-deceleration movement of the head (such as whiplash) can cause postconcussional syndrome." Binder and Rattok[32] described how LOC does not always occur with concussion, and direct blows to the head are not necessary to cause concussions. Instead, concussive effects may be associated with a whiplash injury if the acceleration and deceleration are sufficiently rapid.[32] Lezak,[33] in her recent textbook, wrote that diffuse axonal injury can occur without any direct impact on the head, as it requires only the condition of rapid acceleration/deceleration such as takes place in whiplash injuries. Lezak also noted that the neuropsychological sequelae of concussion without LOC do not differ in severity from those occurring when there is a brief comatose period. Lezak described how patients whose injuries seem mild, as measured by most accepted methods, may have relatively poor outcomes, both cognitively and socially.

In the recent psychiatric textbook *Neuropsychiatry of Traumatic Brain Injury*, McAllister[34] defined mild brain injury as including injury with brief (less than 20 minutes) or no loss of consciousness and with GCS scores, when available, of 13 to 15. McAllister described how "mild" brain injury is a misnomer, because the sequelae may be anything but a minor problem. Instead, the constellation of symptoms that make up postconcussive syndrome leads to a surprisingly high rate of disability.

Finally, Alexander[13] recently reviewed the pathophysiology, natural history, and clinical management of mild traumatic brain injury. He describes how MTBI includes cases in which there is no LOC, but simply a brief period of dazed consciousness.

Mild Traumatic Brain Injury Criteria

Because cognitive impairment may occur without LOC, a more accurate definition for "concussion" needs to be given than that provided in DSM-IV for PCD. Also, instead of the term "head injury," a more appropriate term would be "brain injury," because it has been shown that brain injury may occur without actual damage to the head (e.g., severe whiplash). A definition of MTBI has been developed by the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine[35] (see Table 2). The definition of MTBI also includes the brain undergoing an acceleration/deceleration movement (i.e., whiplash) without direct external trauma to the head. The subsequent symptoms include physical symptoms of brain injury (e.g., nausea, vomiting, dizziness, headaches, blurred vision), cognitive deficits (involving memory, attention and concentration, and executive functioning), and behavioral changes (including irritability, disinhibition, and emotional lability). The authors also described how symptoms of brain injury may persist for varying lengths of time after such a neurological event, and how some patients may not become aware of, or admit, the extent of their symptoms until they attempt to return to normal functioning. In such cases, the evidence for MTBI must be reconstructed. MTBI may be overlooked in the face of more dramatic physical injury (e.g., orthopedic or spinal cord injury).[35]

I suggest that the definition of MTBI would most appropriately provide the minimum criteria for diagnosing a concussion. PCD may also occur in patients with more severe brain injury, but the patients should at least have fulfilled the criteria for MTBI to be given a diagnosis of PCD. Using this definition of concussion would be most consistent with the present literature, which clearly adopts the opinion that LOC does not need to occur, but the patient should at least have experienced an alteration in mental state such as feeling dazed, disoriented, or confused at the time of the accident. Head injury itself or a lengthy period of PTA should not be a requirement for PCD.

Conclusion

This article has examined some of the literature on MTBI and PCD. Although PCD is listed only in an appendix of DSM-IV as a diagnosis that requires further study, the fact that it *has* been included in DSM-IV is significant. Because it has now been defined in DSM-IV, clinicians may use these listed criteria as the basis for making the diagnosis of PCD and providing medicolegal opinions. However, a more appropriate definition of concussion has been suggested in this article. It is clear that LOC is not necessary for PCD to occur. Similarly, a lengthy period of PTA is not required. In the future, precise definitions of terms such as LOC and PTA will be required for diagnosis, research, and communication. The cognitive sequelae of PCD should be expanded to include difficulties with executive functioning and other cognitive deficits, and the duration of symptoms may be less than three months.

Functional imaging techniques such as PET, SPECT, and MRI may soon shed further light on patients who suffer from cognitive deficits following MTBI despite having experienced no LOC.

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